

Amendments to the Claims:

1. (Original) A method for treating a patient having a disease associated with over-expression of EZH2, comprising:
administering to the patient a therapeutically effective amount of a DNA methylation inhibitor.
2. (Original) The method of claim 1, further comprising:
detecting a level of EZH2 expression in the patient.
3. (Original) The method of claim 1, wherein the disease associated with over-expression of EZH2 is selected from the group consisting of: restenosis, benign tumor, cancer, hematologic disorder, and atherosclerosis.
4. (Original) The method of claim 3, wherein the cancer is an early stage cancer.
5. (Original) The method of claim 3, wherein the cancer is a late-stage cancer.
6. (Original) The method of claim 5, wherein the cancer is a late-stage, metastatic cancer.
7. (Original) The method of claim 3, wherein the benign tumor is selected from the group consisting of: hemangiomas, hepatocellular adenoma, cavernous haemangioma, focal nodular hyperplasia, acoustic neuromas, neurofibroma, bile duct adenoma, bile duct cystanoma, fibroma, lipomas, leiomyomas, mesotheliomas, teratomas, myxomas, nodular regenerative hyperplasia, trachomas and pyogenic granulomas.
8. (Original) The method of claim 3, wherein the cancer is selected form the group consisting of: breast cancer, skin cancer, bone cancer, prostate cancer, liver cancer, lung cancer, brain cancer, cancer of the larynx, gallbladder, pancreas, rectum, parathyroid, thyroid, adrenal, neural tissue, head and neck, colon, stomach, bronchi, kidneys, basal cell carcinoma, squamous

cell carcinoma of both ulcerating and papillary type, metastatic skin carcinoma, osteo sarcoma, Ewing's sarcoma, veticulum cell sarcoma, myeloma, giant cell tumor, small-cell lung tumor, islet cell tumor, primary brain tumor, acute and chronic lymphocytic and granulocytic tumors, hairy-cell tumor, adenoma, hyperplasia, medullary carcinoma, pheochromocytoma, mucosal neuronms, intestinal ganglioneuromas, hyperplastic corneal nerve tumor, marfanoid habitus tumor, Wilm's tumor, seminoma, ovarian tumor, leiomyomater tumor, cervical dysplasia and in situ carcinoma, neuroblastoma, retinoblastoma, soft tissue sarcoma, malignant carcinoid, topical skin lesion, mycosis fungoide, rhabdomyosarcoma, Kaposi's sarcoma, osteogenic and other sarcoma, malignant hypercalcemia, renal cell tumor, polycythermia vera, adenocarcinoma, glioblastoma multiforma, leukemia, lymphoma, B-Cell Non-Hodgkin's lymphoma, malignant melanoma, and epidermoid carcinoma.

9. (Original) The method of claim 3, wherein the hematological disorders are selected form the group consisting of acute myeloid leukemia, acute promyelocytic leukemia, acute lymphoblastic leukemia, chronic myelogenous leukemia, the myelodysplastic syndromes, and sickle cell anemia.

10. (Original) The method of claim 1, wherein the disease associated with over-expression of EZH2 is early-stage prostate cancer.

11. (Original) The method of claim 1, wherein the disease associated with over-expression of EZH2 is late-stage, metastatic prostate cancer.

12. (Original) The method of claim 1, wherein the disease associated with over-expression of EZH2 is early-stage non-Hodgkin's lymphoma.

13. (Original) The method of claim 1, wherein the disease associated with over-expression of EZH2 is late-stage non-Hodgkin's lymphoma.

14. (Original) The method of claim 2, wherein the detecting includes detecting the level of EZH2 expression in vivo in the patient.
15. (Original) The method of claim 2, wherein the detecting includes detecting the level of EZH2 expression ex vivo in the patient.
16. (Original) The method of claim 2, wherein the detecting includes detecting the level of EZH2 expression in a sample derived from the patient.
17. (Original) The method of claim 16, wherein the sample is derived from diseased tissue or organ of the patient.
18. (Original) The method of claim 16, wherein the sample is derived from the patient's prostate gland.
19. (Original) The method of claim 2, wherein the level of EZH2 expression is a level of EZH2 mRNA.
20. (Original) The method of claim 2, wherein the level of EZH2 expression is a level of EZH2 protein.
21. (Original) The method of claim 2, further comprising:
comparing the level of EZH2 expression in the patient with a level of EZH2 expression in a control sample.
22. (Original) The method of claim 21, wherein the control sample is obtained from a healthy tissue or organ of the patient.
23. (Original) The method of claim 21, wherein the control sample is obtained from a healthy individual.

24. (Original) The method of claim 2, wherein the detecting is performed prior to the administering of the DNA methylation inhibitor.
25. (Original) The method of claim 2, wherein the detecting is performed post the administering of the DNA methylation inhibitor.
26. (Original) The method of claim 2, wherein the detecting is performed both prior to and post the administering of the DNA methylation inhibitor.
27. (Original) The method of claim 2, wherein the therapeutically effective amount of the DNA methylation inhibitor is determined based upon the level of EZH2 expression in the patient.
28. (Original) The method of claim 1, wherein the DNA methylation inhibitor is a cytidine analog.
29. (Original) The method of claim 28, wherein the cytidine analog is 5-aza-cytidine.
30. (Original) The method of claim 28, wherein the cytidine analog is decitabine.
31. (Original) The method of claim 30, wherein decitabine is administered to the patient via an intravenous infusion at a dose of 2-50 mg/m² a day.
32. (Original) The method of claim 30, wherein decitabine is administered to the patient via an intravenous infusion at a dose of 5-20 mg/m² a day.
33. (Original) The method of claim 30, wherein decitabine is administered to the patient via an intravenous infusion at a dose of 1-100 mg/m² a day.

34. (Original) The method of claim 30, wherein decitabine is administered to the patient via an intravenous infusion at a dose of 1-100 mg/m² a day for at least 3 days per treatment cycle.
35. (Original) The method of claim 1, further comprising:
administering to the patient a therapeutically effective amount of a histone deacetylase inhibitor.
36. (Original) The method of claim 35, wherein the histone deacetylase inhibitor is trichostatin A.
37. (Original) The method of claim 36, wherein trichostatin A is administered to a patient by continuous intravenous infusion for at least 2-3 weeks at a dose of 100-2000 mg/m².
38. (Original) The method of claim 36, wherein trichostatin A is administered to a patient by continuous intravenous infusion for at least 2-3 weeks at a dose of 250-1000 mg/m².
39. (Original) The method of claim 36, wherein trichostatin A is administered to a patient by continuous intravenous infusion for at least 2-3 weeks at a dose of 500-800 mg/m².
40. (Original) The method of claim 35, wherein the histone deacetylase inhibitor is depsipeptide and administered intravenously.
41. (Original) The method of claim 40, wherein depsipeptide administered to a patient by continuous intravenous infusion for at least 4 hours per day for a week at a dose of 2-100 mg/m².
42. (Original) The method of claim 40, wherein depsipeptide administered to a patient by continuous intravenous infusion for at least 4 hours per day for a week at a dose of 5-50 mg/m².
43. (Original) The method of claim 40, wherein depsipeptide administered to a patient by continuous intravenous infusion for at least 4 hours per day for a week at a dose of 5-15 mg/m².

44. (Original) The method of claim 35, wherein the histone deacetylase inhibitor is phenylbutyrate and administered intravenously.
45. (Original) The method of claim 44, wherein the phenylbutyrate is administered to a patient by continuous intravenous infusion for at least 2-3 weeks at a dose of 100-2000 mg/m².
46. (Original) The method of claim 44, wherein the phenylbutyrate is administered to a patient by continuous intravenous infusion for at least 2-3 weeks at a dose of 250-1000 mg/m².
47. (Original) The method of claim 44, wherein the phenylbutyrate is administered to a patient by continuous intravenous infusion for at least 2-3 weeks at a dose of 500-800 mg/m².
48. (Original) The method of claim 35, further comprising:
detecting a level of EZH2 expression in the patient prior to the administration of the DNA methylation inhibitor and the histone deacetylase inhibitor.
49. (Original) The method of claim 1, further comprising:
administering to the patient a therapeutically effective amount of an EZH2 antagonist.
50. (Original) The method of claim 49, wherein the EZH2 antagonist is an antisense nucleic acid, a ribozyme, a small interfering RNA, or a triple helix molecule against EZH2.
51. (Original) The method of claim 49, wherein the EZH2 antagonist is an antibody against EZH2.
52. (Original) The method of claim 49, wherein the EZH2 antagonist is administered prior to the DNA methylation inhibitor.
53. (Original) The method of claim 49, further comprising:

administering to the patient a therapeutically effective amount of a histone deacetylase inhibitor.

54. (Original) The method of claim 53, further comprising:
detecting a level of EZH2 expression in the patient prior to the administration of the DNA methylation inhibitor and the histone deacetylase inhibitor.

55. (Original) The method of claim 53, further comprising:
administering to the patient a therapeutically effective amount of an anti-neoplastic agent.

56. (Original) The method of claim 55, wherein the anti-neoplastic agent is selected from the group consisting of alkylating agents, antibiotic agents, retinoids, anti-metabolic agents, hormonal agents, plant-derived agents, anti-angiogenesis agents, and biologic agents.

57. (Original) The method of claim 55, wherein the anti-neoplastic agent is administered to the patient post the administration of the DNA methylation inhibitor.

58. (Original) The method of claim 55, further comprising:
detecting a level of EZH2 expression in the patient prior to the administration of the DNA methylation inhibitor and the histone deacetylase inhibitor.

Claims 59. -105. (Canceled)